SPINAL CORD FROM A CASE OF POTTS' DISEASE.

By HENRY D. BOYER, M.D.

THE following case was under the care of Dr. J. P. Willits in the Germantown Hospital in the spring of 1894.

Unfortunately he was an Italian who spoke no language other than his own, and his previous history could not be ascertained.

When brought to the hospital he was a middle-aged man, with the objective symptoms of a chronic myelitis. And no direct cause for the cord disease could be found. He had evidently been in bed for some time. There were much emaciation, complete paraplegia, and contractures of the thighs and the legs with fibrous anchylosis at the hips and the knees. The sensation was completely lost from the umbilicus down. The knee jerks could not be tested on account of the contractures. There was full control over the bladder and the bowel sphincters. The arms were in no way affected, except by the general weakness. The back was perfectly straight. There was no point of especial tenderness along the spine, nor any pain in any other part of the body. The temperature was at times hectic, but no focus of pus could be found.

This condition remained much the same for some five months, the symptoms of myelitis becoming gradually worse until death occurred. Towards the latter part of this time, the control of the bladder and the bowels was lost, and a bed sore developed on thesac rum and right heel.

The post-mortem was made by Dr. Burr, who gave me the following notes of it and the spinal cord.

At the time of the man's death, there remained the paralysis and the contractures of the leg as before noted. There was no angularity of the spinal column and no prominence looking like an abscess near the surface. On cutting down on the spine, there was opened a large pocket holding almost a pint of pus.

This was found under and to the inner side of the right scapula. The pus had burrowed into the posterior mediastinum, but not into the pleural cavity. The laminæ of the dorsal vertebræ from the fifth down were carious. The bodies of the same vertebræ were markedly carious and filled with cheesy matter. The most marked destruction of bone was in the fifth, sixth and seventh dorsal vertebræ. The meninges of the cervical portion of the cord were normal. Those of the lumbar region were almost free of deposit. In the dorsal region, the dura was greatly thickened. At the point of most disease, from the fifth to the eighth dorsal vertebra, the cord was completely surrounded by a thickened dura, at some places one-half of an inch in thickness. The brain was normal. The lungs showed miliary tuberculosis with pleural adhesions.

The microscope shows the extreme dural thickness to be due to an inflammatory process in the dura itself, with new cell formation, and to a cellular and a cheesy deposit on the outer side of the dura.

The cord is greatly pressed upon by this new tissue. It is distorted and nearly destroyed at this site by both the mechanical and some inflammatory processes. Secondary degenerative lesions are found both above and below this point. At the place of greatest pressure, there is shown some inflammatory changes. Many new cells are present where only nervous tissue should exist.

There is no distinct line of demarcation between the gray and the white matter. Both being almost totally destroyed. The cells in the anterior horns of the gray matter are few in number. Some are destroyed. Others are not perfect, having lost some of their processes. The central canal is filled by new epithelial cells. The white matter is mostly degenerated in all portions of the cord at this level. Most of the axis cylinders and the myelin sheaths are totally destroyed. Other myelin sheaths are undergoing destruction, being swollen or narrowed, according to the degree of degeneration. Small particles of myelin, not in cylindrical form, are everywhere found through the section. There are a few of both myelin sheaths and axis cylinders that are normal. These are found near the centre of the section, and around the remains of the gray matter where least pressed upon.

The secondary degeneration both above and below the point of most pressure shows the same kind of change as above described,but to a less extent. It follows fixed columns. In none of these is the degeneration so marked as at the transverse myelitis.

In the cervical enlargement, the posterior median tracts of Goll are almost totally destroyed. The columns of Burdock are a very little affected. There is another small degenerated area in the lateral ascending columns. This is close to the margin of the cord and in the extreme lateral part.

A section very high, near the medulla shows the degeneration in the posterior median columns. That in the ascending lateral tract is not so wide as in the cervical enlargement. The other lateral tracts are normal above the lesion.

Below in the lower dorsal region all the lateral columns are involved, except the lateral fundamental zone lying near the anterior gray matter. The direct pyramidal tracts are also marked by the degeneration.

In the lumbar region, the antero-lateral tracts all show more or less destruction, except immediately around the anterior gray matter. The degeneration is greatest near the surface.

Below the lesion the posterior columns are not affected.

I wish to thank Drs. Burr and Willits for the privilege of presenting this case.

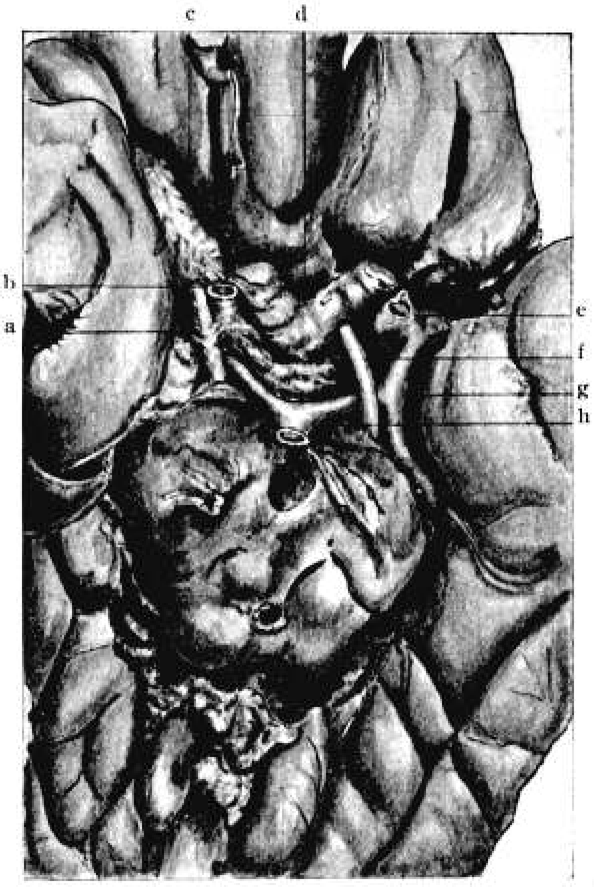

FIG. 1. Drawing showing the gross lesions: a, right third nerve adherent to fibroid mass; b, right internal carotid plugged by a thrombus; c, fibroid mass filling the Sylvian fissure and extending backward to the crus; d, chiasm; e, left internal carotid containing recently organized thrombus; f, left third nerve; g, left postcommunicant at junction with postcerebral; h, basilar giving off postcerebral. *

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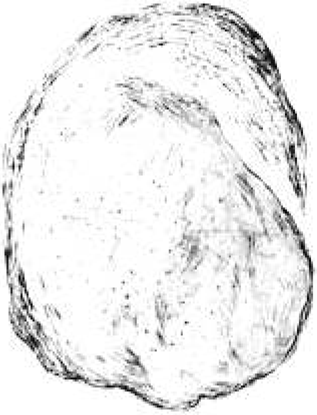

FIG. 2. Section of right oculo-motor nerve, showing complete degeneration, and almost total disappearance of the nerve fibres. Low magnification.

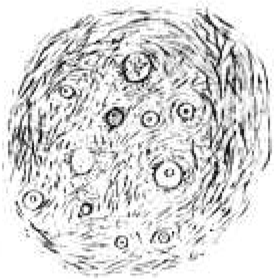

FIG. 3. High magnification of FIG. 2.